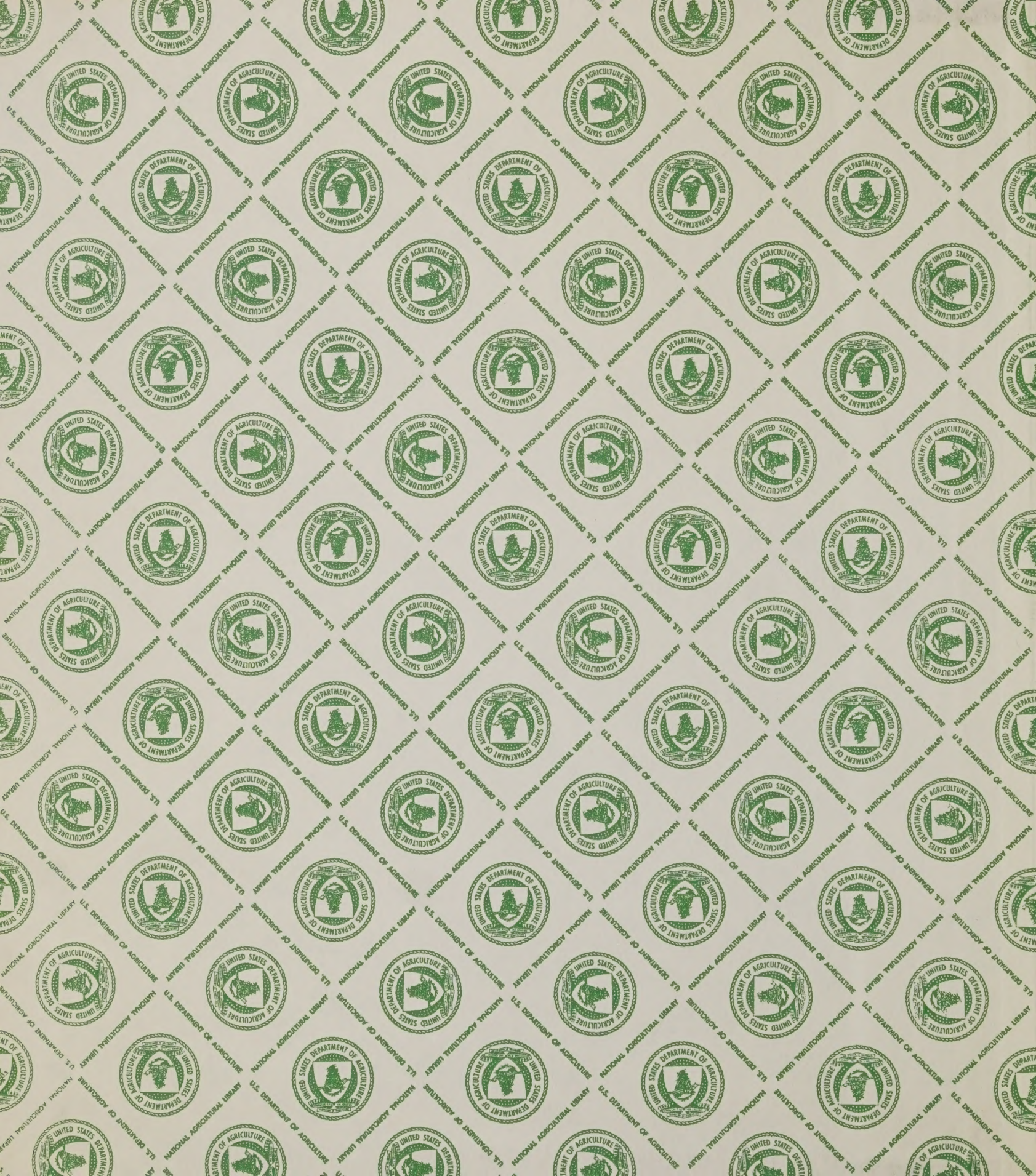
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POTATO SLIMY SOFT ROT.

POTATO: SLIMY SOFT ROT.

Cause: Bacteria (Bacillus carotovorus group).

This disease of the potato belongs to the class of slimy soft rots described elsewhere, but, since it presents itself as a special phase in southern potatoes, a separate discussion is warranted. It is a soft, soupy, exceedingly foul smelling rot. Unless affected tissue is discolored by other causes, there is little change in color, the disintegrated tissue being whitish to yellow. The boundary between the soft disintegrated and the firm sound tissue is very sharp. As the more or less watery tissue dries, it becomes slimy and finally, when completely dry, a mere chalky white crust. During shipment the skin of affected areas is broken easily, and the slime commonly is smeared over neighboring sound tubers. In car-lot shipments this very materially increases the difficulty of sorting.

Slimy soft rot very generally invades killed tissue. In a wet soil, tubers often become asphyxiated, and the bacteria enter the tuber through scab wounds, through lenticels (the breathing pores of the tuber), through broken places in the skin, or through diseased stolons. Infection also occurs very generally through bruises received during digging and handling. If infected tubers are dug while wet, and scalded by exposure to the hot sun, or if sacked with the wet soil adhering, a procedure which may lead to heating or sweating, the rot may progress rapidly from the surface inward through the whole tuber.

Brown rot may be followed by slimy soft rot as a result of which large cavities are formed in the interior of the tuber. The decayed tissue often is so viscous that it can be pulled like very soft taffy.

Slimy soft rot is prevalent as a surface rot in potatoes shipped from Florida, Louisiana and Texas. It is favored by wet, hot conditions in the field, in transit or in storage. After the rot has once started, the temperature is of less importance than the humidity. This rot occurs also in northern potatoes. Tubers affected with black-leg or with scald or freezing injury are predisposed to slimy soft rot. In fact, slimy soft rot commonly follows freezing injury if the temperature after thawing is high enough to permit the growth of the bacteria.

The control measures are careful sorting of stock which has been subjected to unfavorable conditions before it is put into transit or storage, and improvement of handling methods to avoid wounds and scald. The rot progresses and spreads very rapidly in transit and storage, but can be checked by rapid drying of affected stock.

POTATO

HOLLOW HEART GROWTH CRACKS

POTATO: SURFACE MOLD.

Cause: Various fungi.

Under transit or storage conditions, potatoes often show a superficial development of mold growth which is absolutely non-injurious to the tubers except as it affects their appearance. Various fungi are concerned, including blue mold and Rhizoctonia, and their growth is favored by the presence of moisture, especially the juice from nearby frozen or rotted tubers. Wet, dirty tubers and cut or bruised areas are likely to become covered with surface molds.

Surface molds are objectionable in bulk shipments because of the bad appearance of the stock.

Proper ventilation to dry the surfaces of the potatoes will aid in preventing this trouble.

POTATO: HOLLOW HEART.

Cause: Rapid growth.

Hollow heart is a more or less irregular cavity in the center of the tuber, varying in size and often lined by tissue which is browned and glassy.

Hollow heart occurs most frequently in large, coarse, rapid-growing varieties, particularly when these are grown in very fertile soils. It also is common in red varieties grown in the sand hills region of Nebraska.

Hollow heart and black heart may be confused since cavities occasionally develop in tubers affected with black heart; but these differ from those of hollow heart by their association with discolored tissue.

Usually hollow heart cannot be detected until the affected potatoes are cut. It does not affect their keeping quality.

Affected stock is very undesirable for table use.

Ref. (49).

POTATO: GROWTH CRACKS; SECOND GROWTH.

Cause: Unfavorable growing conditions.

Second growth and misshapen tubers are marked by a development of knobs on the tuber's surface, and growth cracks by wide, deep fissures, which are usually covered with the normal protective cork layer of the tuber.

These malformations occur in all varieties and in all localities, but are most pronounced where there is considerable fluctuation in the water supply of the growing crop.

Second growth tissues are immature, and are subject to peeling, bruising, and freezing injury. These knobs are often broken off, opening a way for Fusarium infection.

Growth cracks ordinarily are not subject to tuber rot infection, whereas mechanical injury such as cuts, splits, and bruises, which are due to careless handling, do not develop the normal layer of cork, hence become infected very readily.

Tubers showing these deformities are of inferior grade, since a considerable waste in paring is necessitated.

SECOND GROWTH POTATO

ENLARGED LENTICELS

POTATO: ENLARGED LENTICELS.

Cause: Excessive water supply during the growing season.

This is marked by flat, pimple-like swellings, which occur at the lenticels of tubers. These swellings are formed by proliferation of cells lining the cavity of lenticels. Normally these cells remain dormant but they may enlarge and divide if potatoes are grown in very wet soil or if the soil becomes waterlogged after the tubers are formed. If this process continues long enough a mass of cells protruding above the tuber surface is formed.

At first these cells are quite soft, whitish in color, and quite easily rubbed off. Later they harden and become brown and corky.

Potatoes showing enlarged lenticels are objectionable because of bad appearance of the stock. Their food value is not impaired, since the swellings are superficial.

POTATO: IMMATURITY.

Cause: Premature death of vines in the field, or premature digging of the crop.

Immaturity is characterized by excessive peeling of the skin, by a greenish tint under the skin, and by a tendency to excessive wilting and flabbiness during transit and storage.

This trouble occurs mainly in late varieties of the North and in southern potatoes dug for early marketing.

Immature tubers are subject to bruising, therefore to Fusarium tuber rot infection, to freezing injury, and to excessive shrinkage if they are shipped in overheated cars. They are not high grade stock and often are bitter in taste.

POTATO: NET NECROSIS AND INTERNAL BROWN SPOT.

Cause: Unfavorable field conditions (possibly mosaic disease).

Net necrosis is characterized by fine, extensive brown streaks which follow the vascular tissues and form a network. The discoloration begins at the stem end, where it is often localized; or, in later stages, it may extend throughout the whole tuber. The discolored tissues are dead.

It occurs in many varieties, and is favored by poor soil conditions and by hot weather.

Internal brown spot is marked by brown or yellow regions of dead tissues scattered throughout the tuber. The affected regions may be small or large, even one-half inch in diameter. They may be scarce or numerous, and are isolated and not connected as in net necrosis.

The disease occurs in all varieties, and is favored by an inadequate water supply during the growing period.

Neither net necrosis nor internal brown spot should be confused with freezing injury, which sometimes produces very similar symptoms, but usually is otherwise distinguishable.

These internal discolorations are not decay and do not impair the food value of the tuber, but affected portions usually are rejected in the preparation of potatoes for the table.

Ref. (49); (50).

POTATO: FREEZING INJURY.

Cause: Exposure to low temperatures in the soil or during harvest, transit, or storage.

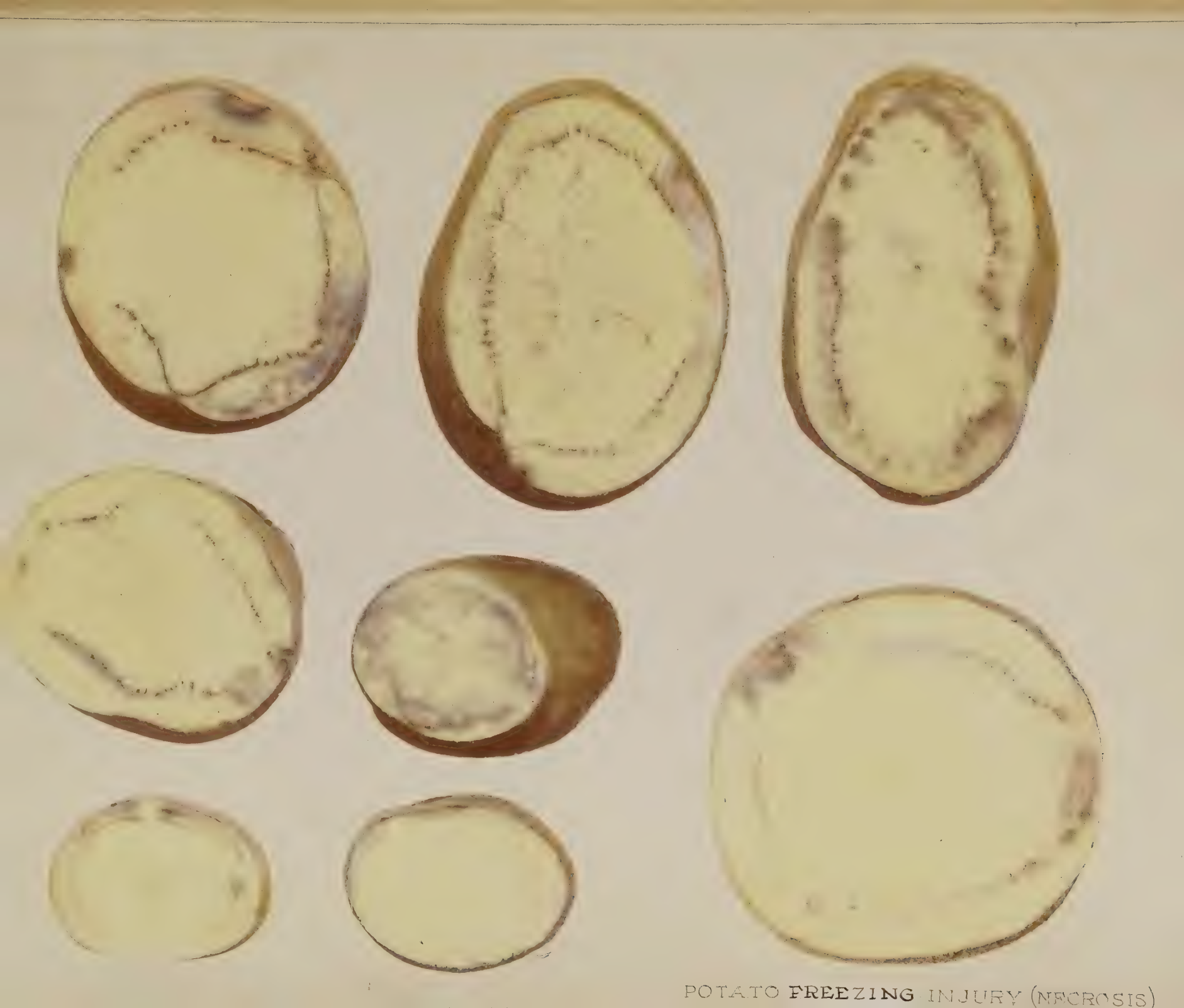
The symptoms of freezing injury are varied and complex and may be general, appearing in all the tissues of a tuber, or local and restricted to sharply limited regions of the tuber. They depend upon the variety, maturity, and individuality of the potato affected, upon the varying predisposition of the several tissues of the tuber to freezing injury, upon the temperature to which the tuber was exposed, and upon the rate of fall of the temperature as well as the duration of the exposure. The symptoms also depend upon the interval between the time of injury and examination of the affected tuber, and may or may not depend upon the rate of thawing and the conditions under which this takes place.

Thoroughly frozen potato tissue no longer possesses the natural crispness or brittleness of the sound potato. Frozen tissue looks dull and does not cut readily nor with snap. This is due to the formation of ice in the tissues. In the freezing process, the cell water passes out of the cells into the intercellular spaces, the spaces between the cells, where it forms ice crystals. The extent to which the cell water leaves the cells depends upon the point to which the temperature is lowered, upon the rate at which this takes place, upon the duration of the exposure, and upon the nature of the potato.

The symptoms presented by thawed potato tissues are very perplexing. Sometimes in case of severe freezing, the tissues become turgid, blister-like, and swollen, and the skin may be discolored. At other times they are quite firm and remain intact and, contrasted with healthy tissue, merely appear dull and colorless. If only a portion of a tuber was severely frozen, usually a purplish band marks the border of the uninjured tissue. When such a tuber is cut, the border between healthy and injured tissue generally is marked by a sharp purplish to dark brown line. However, this line is not always present.

If no infection sets in, the more or less watery thawed tissue dries down to the consistency of a rather moist mealy mass or a shrunken, dull, grayish, very tough and leathery granular mass which is composed of shrunken cells and starch. Very frequently bluish to black colors develop. In this stage the dried-out starchy remnants of the tissue are separated from the uninjured tissue by a dark brown corky layer.

Very frequently the starchy or corky layer is not formed before infection with Fusarium takes place, or it is subsequently broken and admits the fungus. Severe freezing injury generally affects all tissues straight across the tuber, while Fusarium rot develops most rapidly in the pith of the tuber, leaving a shell of cortical tissue. This characteristic



POTATO FREEZING INJURY (NECROSIS)

sometimes is of service in differentiating freezing injury and Fusarium tuber rot.

Thawed tissue, especially when in a warm damp atmosphere, is predisposed to slimy soft rot. In fact, most severely frozen potato tissue is dead and sooner or later becomes infected with the bacteria which cause this rot. The bacteria get into the watery tissue, which is an excellent growing medium for them, through breaks in the skin, and cause a slimy, mushy, or soupy rot which has a very offensive odor. Frequently the much wrinkled skin of such tubers bursts, and the neighboring potatoes are smeared with the slimy disintegrated tissue which dries out grayish and chalky when exposed to the air.

It is very difficult at times to differentiate freezing injury from injury due to scalding. This is especially true when the first or last freezes of the season occur.

The severe general type of freezing injury is marked by a killing of all the cells of the potato. The less severe type, popularly known as chilling, is definitely localized and restricted to the most susceptible tissues. It is known as freezing necrosis. It appears first in the stem end, and at times only the conducting tissues are killed and appear yellowish-brown to black. Very often the conducting tissues in the pith also are killed and appear as a net resembling net necrosis. The storage tissues also may be affected, either in extensive regions, or in small pockets. Starchy tissues so affected are grayish to steel blue, even black in color, granular, and very tough and leathery.

Sometimes the thawing tissues are pinkish to red in color when freshly cut. These colors change quite rapidly in the air, becoming gray, brown, or dirty red. These colors should not be confused with the pinkish color of the vascular ring or pith tissue of some of the red varieties of potatoes. At times white potatoes which have not been exposed to low temperatures also have a pink color. Potato tissues when freshly bruised and exposed to the air often are reddish in color.

Potatoes subjected to freezing temperatures may show none of the symptoms enumerated above but only a tendency to excessive wilting and flabbiness. The cut tissue may appear glassy and abnormally moist. When cooked they may turn black in spots or may have a sweet taste. This condition also is frequently referred to as chilling injury. When potatoes that have become sweet following exposure to low temperatures are kept at normal temperatures the sweet taste disappears. This is due to the fact that the increased respiration due to rise in temperature oxidizes the excess of sugars which accumulated at temperatures of 29° to 34° F. At these tempertures the rate of respiration is cut down more than the rate of digestion of starch into sugar and consequently the sugars accumulate.

POTATO FREEZING INJURY.

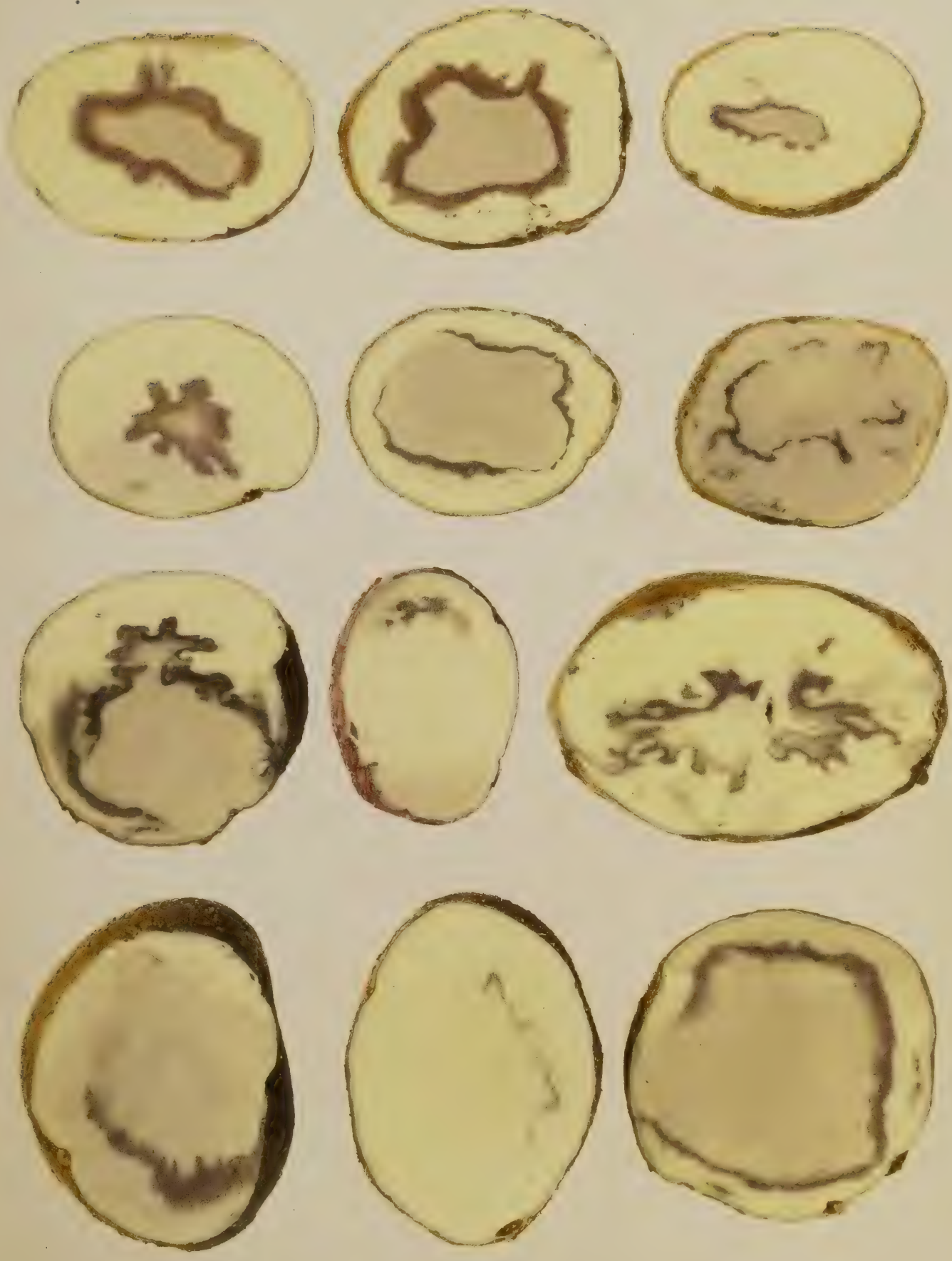
It is very difficult at times to differentiate immature tubers from those showing the less severe types of freezing injury. In both cases the tissues may have a greenish yellow color, and may be flabby and bitter in taste. Immature tubers are also bruised easily by impact or pressure, which causes regions in the tuber to resemble chilled tissue so closely that a diagnosis is excedingly difficult. It also is exceedingly difficult to differentiate between thawed, dried-out tissues and bruised tissues.

It seems in fact that the presence of ice in the tissues is the only positive proof of freezing injury. All other characteristics enumerated as symptoms of freezing and chilling injury can also be brought about by other causes.

Slightly affected stock without extensive, discolored, killed regions is suitable for food even though it is not as palatable as the uninjured. It shrinks excessively in transit and storage.

Guarding against the exposure of potatoes to temperatures under 32° F. will prevent this type of injury. Losses during transit may be avoided by careful sorting to eliminate field frozen tubers previous to shipment, and the use of all proper precautions to prevent freezing injury during hauling, loading, and rail shipment. This involves proper lining and preheating of the car, installation of false flooring and ends, and an adequate heating apparatus regulated during transit so as to avoid both freezing and black heart injury.

Ref. (33a).



POTATO BLACK HEART.

POTATO: BLACK HEART.

Cause: High temperature or insuffcient aeration.

The symptoms of black heart vary, depending upon whether the potatoes are exposed to high temperatures with an adequate air supply, or to high or normal temperatures with an insufficient air supply. In the former case, no external symptoms develop; in the latter, both external and internal symptoms appear.

The external symptoms of black heart are moist areas on the surface which may be purplish at first but turn brown and black in a short time. The internal symptoms are a dark gray to purplish discoloration which later becomes jet-black. The discolored areas are usually sharply set off from the healthy tissue.

Generally the discoloration is restricted to the heart of the tuber, but frequently it radiates to the exterior as well. It may also appear on one side of a potato if this was exposed to a stove in a railroad car or in the storage house. The discolored regions may appear in zones in the peripheral parts and may be absent or less evident in the center. In advanced stages, the affected tissue dries out and forms cavities. In case cavities develop, these may be differentiated from true hollow heart by their black lining of killed tissue.

Exposure of potatoes in the field or in storage or transit for a day to temperatures of 90° to 104° F. causes this injury. Potatoes lying in a very hot soil or lying on the soil after digging may show it. More generally the injury is caused in stove-heated cars or by storing potatoes at 45° to 50° F. in piles deeper than 6 feet. In stove-heated cars, the injury usually is most severe near the stove and at the top of the load.

Black heart injury predisposes affected tissues to slimy soft rot. The heated, killed and watery tissues offer excellent growing conditions for bacteria.

Black heart does not impair the food value of non-affected tissues.

Prevention of this injury in cars involves proper ventilation, protection of the load near the stove with tin sheeting, and careful attention to the fire. The temperature should never go over 60° or 70° F.

The injury can be prevented in storage places by providing proper ventilation and by storing tubers in piles not deeper than 6 feet.

Ref. (1); (68).

POTATO SCALD

POTATO: SCALD.

Cause: Exposure to the hot sun.

Scald on potatoes may appear first as large blisters which soon become sharply depressed or as slightly bleached areas with a very irregular and lobed margin of a darker metallic color. Upon cutting such a tuber, it is found that a shallow surface layer of tissue has been killed. This killed tissue is dull gray in color, and is separated from the uninjured tissue by a brown and very irregular line. The killed tissue, if not infected, dries out and becomes tough and leathery.

In many cases, more of the tuber is involved, both in surface area and depth of the killed tissue. This type has been tentatively named "deep scald," and shows much more discoloration than the ordinary scald. Bluish black patches often surround the lenticels, and the killed tissue darkens very rapidly when cut surfaces are exposed to the air.

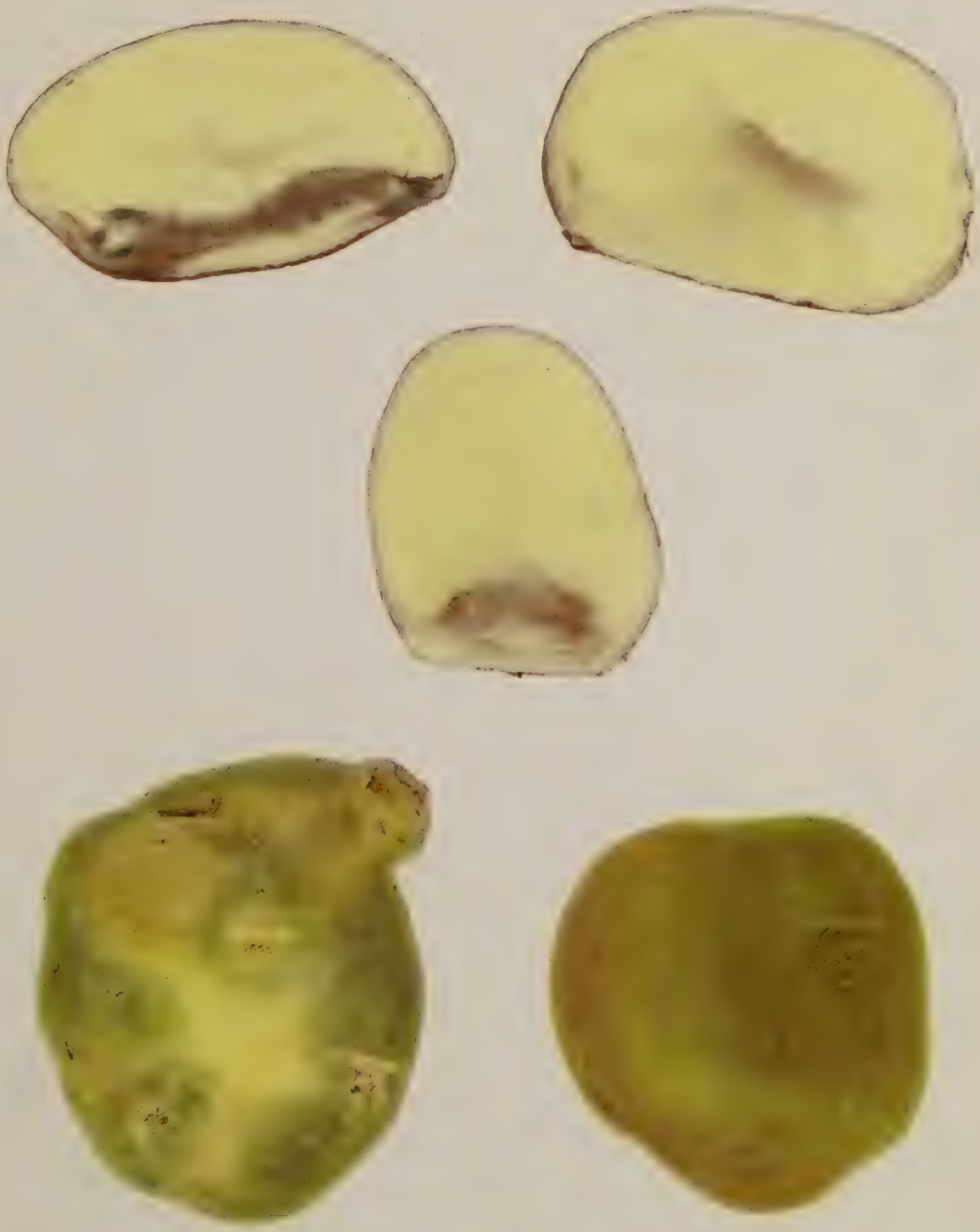
Scald differs from late blight tuber rot in that the affected tissue is not brown in color, and in the sharp line of demarcation between the dead and the living tissue. Scald differs from freezing injury in its much more irregular surface outline and in the more irregular plane of separation between killed and sound tissue. At certain seasons, it may be very difficult to make this differentiation positive without rather careful inspection of car lots. Freezing injury, however, should be accompanied by other unmistakable symptoms.

Scald of potato is probably caused by exposure of newly dug tubers to the hot sun in the field.

Scald is of the highest importance from the market point of view. The killed tissue is very subject to the attacks of rot-producing organisms such as Fusarium and especially the bacteria of slimy soft rot. In the Chicago market in the summer of 1918, there were enormous losses due to slimy soft rot following scald in shipments of potatoes from the South and the entire Mississippi Valley.

It is not advisable to ship potatoes showing scald because of their extreme predisposition to slimy soft rot during transit.

POTATO DEEP SCALD



POTATO SALT INJURY
SUN BURN (BELOW)

POTATO: SALT INJURY.

Cause: Contact of potatoes with the floor or walls of a car in which fertilizers, salt, or salted hides have been shipped.

Usually the side of the potato in contact with the floor or wall is flattened, and the injured tissue is soft and flabby and at times semi-watery. At times the injured tissue is firm and leathery. Before the air has free access to the tissue it usually is colorless. After a short exposure to the air if not infected the injured tissues become brown to black, and begin to dry out. The exterior of the injured region generally is black. Frequently the injured tissue has a distinct salty taste, and salt injury may be thus diagnosed.

Tubers affected with salt injury are predisposed to slimy soft rot, and generally become infected and soupy if not removed promptly from the car.

POTATO: SUNBURN.

Cause: Exposure of tubers to light, either in the field, or during harvest, shipment and storage.

Sunburn is marked by a green discoloration of the surface layers, and in severe cases, of the interior portions of the tuber.

Sunburn occurs in potatoes of all varieties upon prolonged exposure to the light. It is common in eastern stock shipped in midsummer.

Affected stock is bitter and unfit for food.

POTATO: BRUISES, CUTS, AND SPLITS.

Cause: Mechanical injury.

Bruises may be superficial or internal. The most common type of bruise occurs in immature potatoes whose tissues are very tender and crisp, and consequently are easily broken either by pressure or by friction. Often an immature tuber is gouged by rubbing against the container or by being walked upon. In either case, the result is a loss of water by the injured cells, leaving a solid, grayish, starchy deposit. At times this deposit is black, and externally the bruised areas often appear black. These types of bruising are very common in potatoes grown under irrigation. The injury due to gouging is more severe than peeling or feathering though it usually is due to the same causes.

Often potatoes are bruised or crushed by impact or by the weight of the load. In addition to the injuries above described, impact or pressure may cause symptoms which may be wholly internal or may be indicated externally only by small, slightly sunken spots. At times these internal injuries resemble those due to freezing and consist of small pockets of grayish, starchy masses, the remnants of ruptured, dried-out cells.

Most of the cuts in potatoes are caused by digging implements, and by the pernicious custom of handling potatoes with steel shovels or forks. Cuts may be broad and shallow or very deep.

If the cut or bruised tissue has an opportunity to dry out rapidly the injury may be sealed by a grayish granular deposit of starch, and later by a brown layer of cork. Very often, however, especially under moist conditions, Fusarium gets a foothold before the bruised or cut tissue is healed. It may also enter through subsequent breaks in the dry starch layer. In very hot weather, especially in southern potatoes, slimy soft rot gets a start in bruises and cuts. Shallow, broad, or smooth cuts are less subject to infection than those which are deep and ragged.

Potatoes often are fractured by pressure or by impact. Careless throwing about of potatoes, heavy loading, walking upon potatoes, and similar rough treatment, are responsible for these fractures or so-called splits. These may be visible externally or they may be wholly internal. In either case the surfaces of the fracture are lined with a deposit of starch or a brown layer of corky tissue. At times freshly dug potatoes develop splits if they dry out too rapidly.

Bruised, cut, or split potatoes are suitable for food, though the injuries necessitate very appreciable losses in paring. Their presence, however, lowers the market value of a shipment.

These mechanical injuries generally are ports of entry for fungi and bacteria which cause tuber rots. In most cases of Fusarium tuber rot, the infection starts in such injuries. In

SEALED WITHOUT INFECTION WITH INFECTION

POTATO MECHANICAL INJURY.

the early part of the shipping or storage period, it is impossible without a cultural test to determine whether a mechanical injury will remain merely an injury or develop a tuber rot. However, as the season progresses, the rot symptoms become more pronounced.

This type of injury with its frequently disastrous consequences could be prevented if it were constantly borne in mind that potatoes consist of living cells and should not be handled like stones.

POTATO: FLEA-BEETLE DISEASE.

Cause: The larva of the flea beetle (Epitrix cucumeris Harr.).

In the simplest cases flea-beetle injury is marked by corky pimples or raised places on the tuber. At times the surface of such pimples is slightly broken. In the more complex and advanced stages the surface of the tuber is a mass of pimples and of channels or furrows formed by breaking of the skin. The margins of such furrows and channels frequently are elevated and swollen so that the whole furrow stands above the surface of the tuber. This type of injury frequently is known as worm tracks.

By cutting into the pimples or furrows brown, tough splinters of corky tissue are found extending perpendicularly into the tuber tissue. Sometimes these splinters are a fourth of an inch in length. Their presence serves to differentiate the simple, pimple stage of flea-beetle injury from slight nematode injury, and the furrowed type from other tuber diseases such as scab, grub injury, or wire-worm injury.

Flea-beetle injury is most frequent in Colorado potatoes. It injures the appearance of potatoes and generally necessitates very deep paring.

POTATO: NEMATODE DISEASE.

(See Nematode Disease).

RADISH BLACK ROOT.

RADISH: BLACK ROOT.

Cause: A fungus (Rheosporangium aphanidermatum).

This disease of radish roots is characterized by blackened regions of varying size which may be rather superficial, or may extend deeply into the root. The darkened tissue is firm and not disintegrated. The lesions are often cracked or fissured on the surface as a result of root growth, and may be accompanied by some disfiguration of the root due to checked growth.

This disease is of widespread occurrence. Both red and white varieties are susceptible. The fungus persists in the soil and often causes damping-off of seedlings.

Badly affected roots often are found in the market, but are not considered edible, and their presence lowers the value of the bunch.

Control involves crop rotation or soil sterilization. Badly affected roots should not be sent to market.

Ref. (16).

RADISH

WHITE RUST

DOWNY MILDEW

RADISH: MISCELLANEOUS DISEASES.

DOWNY MILDEW.

Cause: A fungus (Peronospora parasitica).

Downy mildew causes angular spots on the leaves, bounded by the larger veins. These spots are yellow in color when viewed from above, and the under surface is covered with a white felt-like outgrowth of mold.

This disease often is found in the market but is of no importance since the root is not attacked.

WHITE RUST.

Cause: A fungus (Cystopus candidus).

The symptoms of this disease on the leaves are small, irregular lesions bearing on the under surface smooth, pure white, porcelain-like cushions or blisters. These rupture, allowing the spores to escape, and the lesions are then recognizable by the torn edge of the empty pustule. Often these lesions cause marked distortion in the leaves and other affected parts.

Since this disease does not occur on the roots, it is of no importance in the market.

ROOT ROT.

Cause: A fungus (Rhizoctonia).

This disease is marked by a rather large brown circular lesion on the side of the root in which the tissue is rotted as in watery soft rot.

This has been found occasionally in the market.

NEMATODE DISEASE.

(See Nematode Disease).

RADISH SLIMY SOFT ROT OF LEAVES.

RADISH; SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

RUTABAGA - DRY ROT

RUTABAGA: DRY ROT.

(See Radish Root Rot).

RUTABAGA: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

RUTABAGA: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

RUTABAGA: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot).

RUTABAGA: NEMATODE DISEASE.

(See Nematode Disease).

SQUASH RHIZOPUS ROT

SALSIFY: WATERY SOFT ROT; SCLEROTINIA ROT.

(See Watery Soft Rot and Beet Watery Soft Rot).

SPINACH: SLIMY SOFT ROT; BACTERIAL ROT.

(See Slimy Soft Rot).

SQUASH: RHIZOPUS ROT.

(See Rhizopus Rot).

SWEET POTATO BLACK ROT.

SWEET POTATO: BLACK ROT.

Cause: A fungus (Sphaeronema fimbriatum).

Black rot is characterized by greenish to nearly black, circular, depressed spots, varying in size from ½ to 2 inches in diameter. At times the spots are very irregular, occuring in bruises and injured places. The lesions are shallow, rarely penetrating to the heart of the root, and the affected tissue is quite firm and tough. Often at the center of the spots small, black bodies are developed, which appear at times as dense masses of small, black bristles. These are the fruiting bodies or pycnidia of the fungus. Wetting the surface of the lesion will bring out better these distinguishing characteristics.

Black rot is of common occurrence in all sweet potato regions. The roots become infected in the field, often through wounds. The diseased areas may enlarge in storage or transit, where overheating and lack of ventilation favor the progress of the disease. Generally, affected stock is culled out and is not sent to the markets.

Affected stock is unfit for table use since the diseased tissue is very bitter. When cooked, the entire root has a bitter flavor. Diseased sweet potatoes also are unsuitable for seed purposes, since black rot is spread by planting infected seed roots.

Ref. (24); (27); (70).

SWEET POTATO: SOIL ROT.

Cause: A slime mold (Cystospora batata).

The symptoms of this disease are small, circular, sunken spots about one-half inch in diameter in which the tissue has dried out and cracked. The lesions may appear as circular pits or depressions from which the diseased tissue has fallen out. There may be some constriction of the root where such lesions are located.

Infection occurs in the field and the disease does not progress in storage.

The economic importance of soil rot is due to the fact that it reduces the yield and disfigures the roots.

Ref. (69).

SWEET POTATO: SCURF.

Cause: A fungus (Monilochaetes infuscans).

Scurf is marked by small, circular, dark clay-colored spots on the skin of the root, which may unite to form large blotches. The spots are only skin-deep. In advanced stages, the skin turns to a deep brown color and becomes wrinkled and rough.

Scurf occurs in practically all sweet potato growing regions.

Infection takes place in the field, but the spots may develop in storage. The disease does not spread from one root to another. The market value of diseased stock is slightly reduced because of its unsightliness.

Ref. (24); (27).

SWEET POTATO - SOFT ROT

SWEET POTATO: SOFT ROT.

Cause: A fungus (Rhizopus nigricans).

This rot is one of those elsewhere described as Rhizopus rot. Soft rot develops very rapidly, and unless checked, soon involves the whole root. In the early stages, the affected tissue is soft, watery, and stringy, and yields a brownish-yellow liquid when compressed. The early stage is also attended by an odor of vinegar or acetic acid. Under warm, moist conditions, the characteristic mycelium and sporangia may appear on the outside of the root. The affected tissue eventually becomes dry, shrunken, and yellowish brown. At this stage an aromatic odor may be detected.

Infection, which takes place through wounds, occurs either in the field, in transit, or in storage and is favored by high temperature and humidity. The rot progresses and spreads very rapidly in transit and storage. Infection may set in at the end or the middle of the root. In the former case, it is called soft rot; in the latter, if encircling the root, ring rot.

Control consists of careful handling of the roots to avoid wounds, careful sorting, proper curing, and storage in a dry atmosphere at a temperature of 50° F. When wet weather prevails during harvest, the shipment of roots not properly dried often results in severe losses. An additional control measure is the disinfection of storage houses and cars with copper sulphate or formaldehyde.

Ref. (24); (27).

SWEET POTATO: STEM ROT.

Cause: Fungi (Fusarium batatatis and F. hyperoxysporum).

In stem rot the end of the root is light and shriveled, and the interior has a deep brown color and a peculiar cinnamon odor. Usually, however, stem rot is not plainly visible externally, and the disease manifests itself merely by a discolored ring, and at times by a secondary ring, visible in transverse sections of the root. If the outer part of the root is carefully removed, the discolored parts appear as a network of brown fibers surrounding the heart.

Stem rot is of widespread occurrence in all sweet potato districts.

This disease causes a wilt of vines in the field, and reduces the yield. Infection of the roots takes place in the field, and the disease progresses only slightly in storage. Stem rot is found infrequently in the market, and is described here principally to avoid confusion with freezing injury, which it somewhat resembles.

Ref. (24).

SWEET POTATO: MISCELLANEOUS DISEASES.

DRY ROT.

Cause: Fungi (Fusarium species).

This disease is comparable to the Fusarium tuber rot of the Irish potato. It is marked by shrunken areas showing discolored flesh, and usually starts at one end of the root. Dry rot is not common or of much importance.

FOOT ROT.

Cause: A fungus (Plenodomus destruens).

This is a storage rot of some importance. It follows a blighting of plants in the field. It is a firm brown rot with pimple-like protuberances on the surface. Foot rot may enter roots through wounds and bruises and thus cause some loss in storage.

Ref. (27).

NEMATODE DISEASE.

(See Nematode Disease).

TOMATO ANTHRACNOSE

TOMATO: ANTHRACNOSE.

Cause: A fungus (Colletotrichum phomoides).

In its early stages, anthracnose is characterized by small, circular, sunken spots of the same color as the sound tissue. These lesions enlarge rapidly, become watersoaked and more sunken, and bear pinkish spore heaps which often coalesce to form a slimy layer. These spore heaps or acervuli turn black at times and resemble the pycnidia of Phoma, so that anthracnose may be mistaken for Phoma lesions. However, the spore heaps never are as definitely rounded as the pycnidia, and the pycnidia never are pink. The centers of anthracnose lesions are at first light brown, and do not become as black and charry as those of Phoma rot.

Anthracnose is common on northern-grown tomatoes, especially New Jersey stock. Green fruit is rarely affected.

The original infection takes place in the field. The disease develops in the field, and may also spread and develop in transit and storage. Often this fungus destroys the entire fruit.

It is advisable not to pack or ship infected fruit because it rots rapidly and is a menace to healthy fruit.

Ref. (30).

TOMATO BUCKEYE ROT

TOMATO: BUCKEYE ROT.

Cause: A fungus (Phytophthora terrestria).

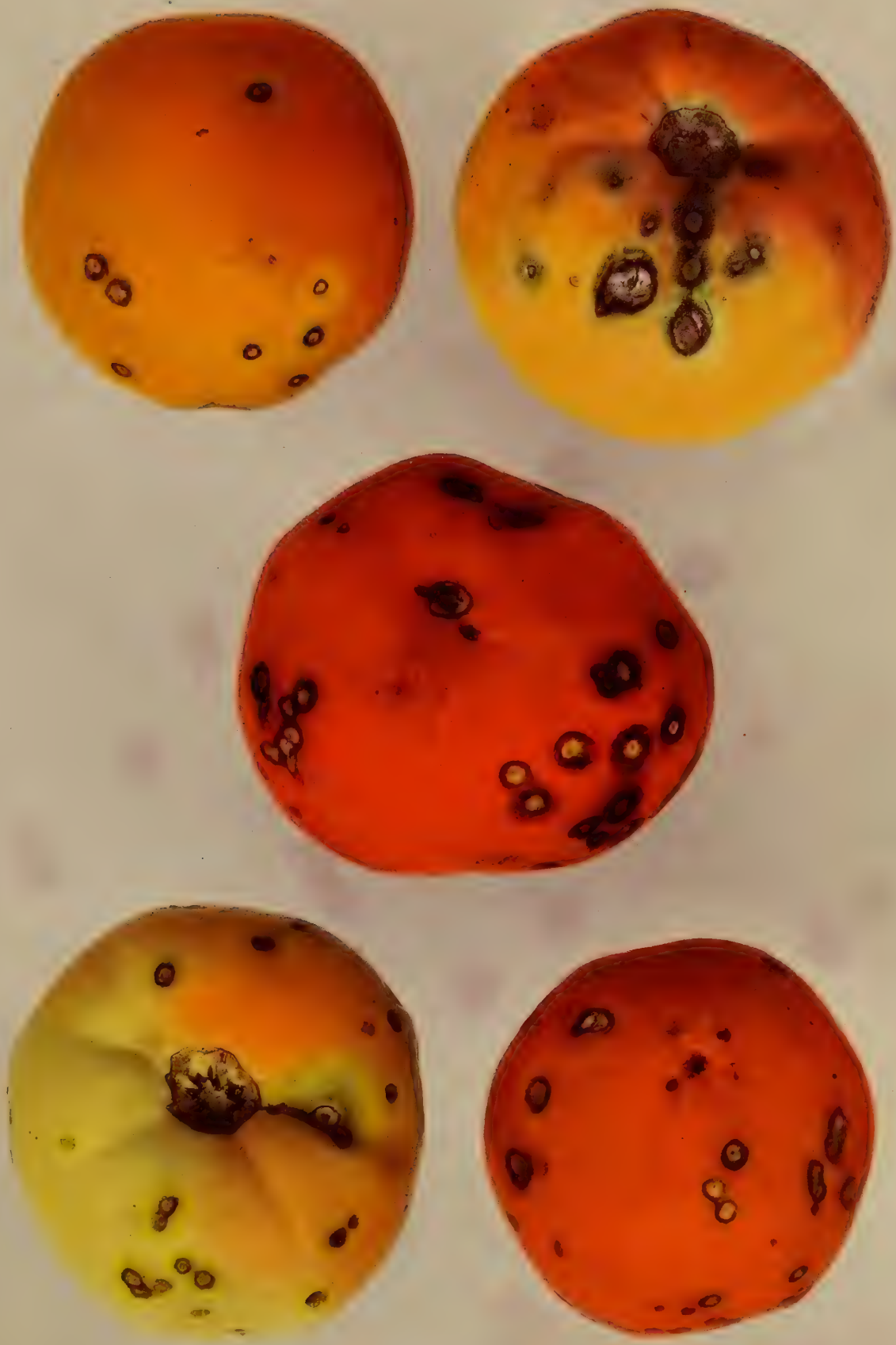
No better description of this disease can be given, so far as the appearance of the fruit on the vine or in transit is concerned, than is implied by the name "buckeye." In color and in its surface appearance, the diseased tissue resembles a horse chestnut very closely. The color is grayish to brown, and the surface is uneven, being sunken in places. The rot at first is firm and even hard. The border is irregular and not sharply defined, and often merges into water-soaked, slightly bleached, green tissue. At times there appear widely separated concentric rings of more or less irregular wavy outline. These rings, however, are by no means a constant accompaniment of the disease. Under moist conditions the lesions generally are covered by a white, fluffy growth of mold.

In case concentric rings are found in buckeye rot, they are farther apart and less regular in outline than in soil rot. In soil rot, the mycelium is brown and may produce sclerotia, while the mycelium of buckeye rot is white and produces no sclerotia. The absence of white, glistening or black sporangia differentiates buckeye rot from Rhizopus rot.

This disease occurs most commonly in southern tomatoes during rainy weather. The original infection takes place in the field, and occurs only on fruit which has been in contact with the soil or close enough to it to be spattered during rains.

As in the case of soil rot, buckeye rot can be controlled in the field by staking the vines to keep them off the ground. At harvest it is advisable to sort out diseased fruit because it is unsuitable for food and is a menace to healthy stock since the fungus passes through the wrappers from diseased to healthy fruit.

Ref. (56); (59).



TOMATO NAILHEAD SPOT.

TOMATO: NAIL-HEAD SPOT.

Cause: A fungus (Alternaria solani).

In the early as well as the later stages, nail-head spot is marked by small, dry, slightly though abruptly sunken scab-like spots which are brown to black in color.

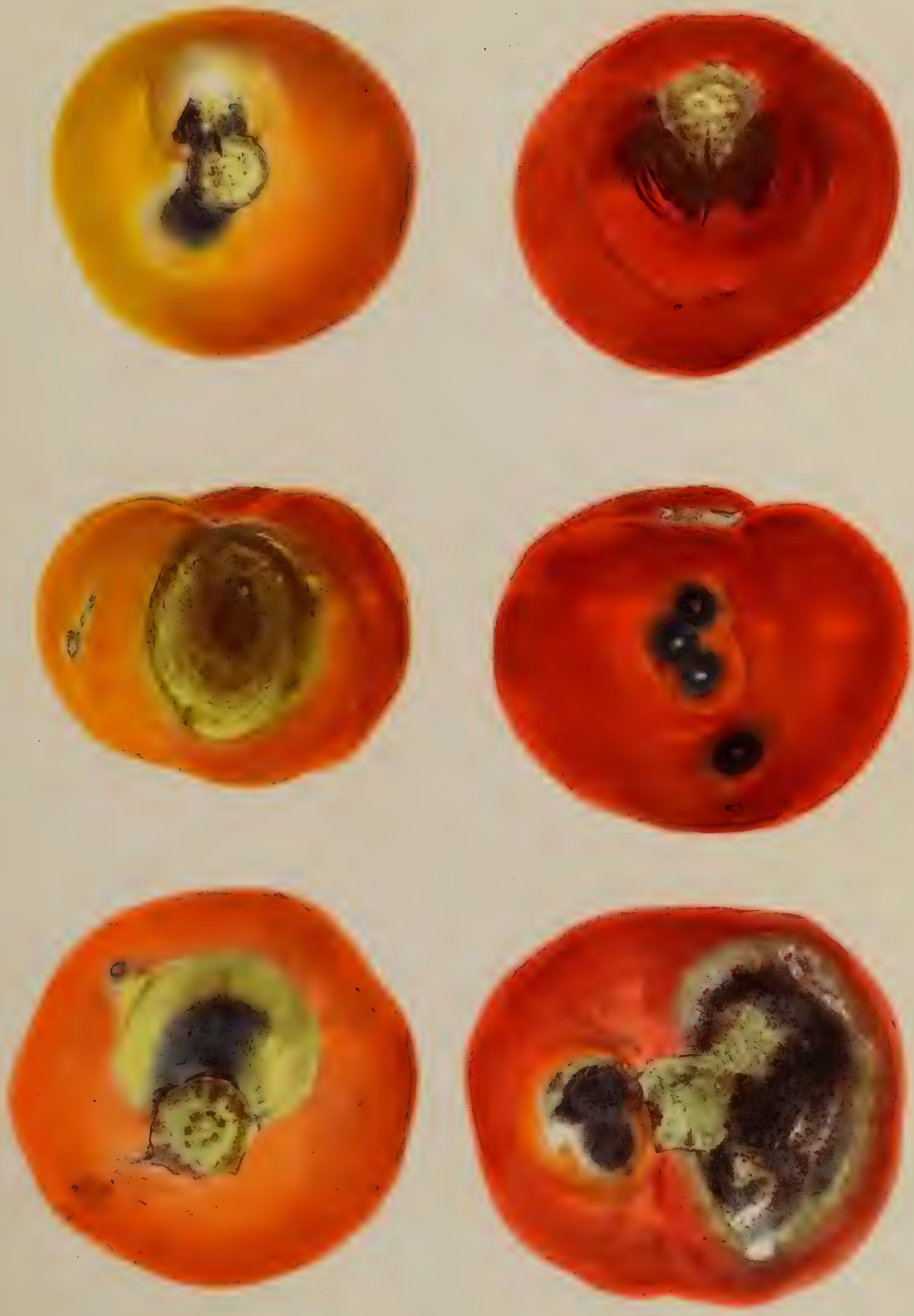
These spots or lesions generally are mere blemishes, and cause little damage. The lesions may be circular or elliptical and at times may coalesce to form a large, irregularly shaped area. At times the spots on ripening fruits are surrounded by a green zone due to delayed ripening of the bordering tissue. The tissue underlying the spot is not affected, and the diseased tissue can easily be removed from the sound tissue underneath. As the lesion ages, it often cracks and opens the way for the entrance of secondary rots such as Phoma.

The small size and the slightly sunken, flat and scab-like nature of the lesions of nail-head differentiate these from the lesions of other tomato fruit diseases, most of which, in contrast, are large or water-soaked, often deeply sunken and involve more rotting of the tissue.

Nail-head spot is a warm-weather disease, and is prevalent on fruit from the Gulf States. It is a serious field disease, attacking leaves, stems and fruits, and is known by the growers as early blight. The fruit is infected before it leaves the field, and the spots are visible when the fruit is packed. The spots may enlarge slowly in transit and storage.

Spraying will control the disease in the field. In packing it is advisable to sort out spotted fruit because its presence lowers the market value of a shipment and affected fruits are predisposed to rots.

Ref. (56); (60).



TOMATO PHOMA ROT

TOMATO: PHOMA ROT.

Cause: A fungus (Phoma destructiva).

In its early stages, Phoma rot is marked on the green fruit by minute, slightly sunken circular spots with a brown or black border and a lighter center. Later the center of the spot becomes covered with black pimple-like bodies. These are known as pycnidia (singular pycnidum). In the early stages, spots on ripe fruit are water-soaked and concave. Later, as they enlarge, the centers become brown to charry black, leathery and firm, and covered with pycnidia. The borders are slightly sunken and water-soaked. The lesions increase indefinitely and quite rapidly in size but remain more or less circular and concave.

The lesions generally occur at the stem end of the tomato, and the disease is often known as stem-end rot. The spots, however, are not confined to this part of the fruit, as the fungus may attack the fruit wherever it is bruised or injured. The fungus may also enter through nailhead spots. Until the lesion becomes very large, the underlying brown or black tissue is quite firm.

Without a microscope it is quite difficult at times to differentiate the various rots of mature tomato fruits caused by Phoma, Colletotrichum, and other fungi. This is especially true when secondary rots have set in and covered up or confused the typical clear-cut symptoms.

It appears, however, that anthracnose and other fruit rots are more common on over-ripe fruit, and occur in southern stock later in the season than does the Phoma rot. The slimy, orange-pink spore masses of anthracnose aid in differentiating it from Phoma rot. In Phoma rot, slimy spore masses appear very frequently at the openings of the pycnidia, but these are whitish rather than pink in color.

This disease is most marked in southern winter-grown tomatoes; that is, the Cuba, Florida, and California crops. It was exceedingly destructive to Cuban tomatoes during the winter of 1917-18, and to Florida tomatoes during April and May of the same season.

The disease occurs on the vines, where it is known as Phoma blight or black spot. It is not known definitely whether the original infection of the fruit destroyed in transit occurs in the field or in the packing house. The disease develops in transit and in the ripening rooms, and is favored by a warm, moist atmosphere.

Affected fruit is subject to infection with bacteria and other fungi.

No satisfactory control of the disease in transit and in the ripening rooms has been worked out.

Ref. (32); (56); (60).

TOMATO SOIL ROT

TOMATO: SOIL ROT.

Cause: A fungus (Rhizoctonia).

In its early stages, soil rot on green or ripe fruit is characterized by firm, brown spots which may be marked with concentric rings. Later they increase in size and generally a brown, firm mat of mold covers the older lesions. On mature fruit in the market, soil rot lesions are large, brown, water-soaked areas not sunken and not concentrically marked.

Soil rot progresses and spreads very rapidly in transit and storage, the fungus passing from one fruit to another through the wrappers. The most striking characteristic under these circumstances is the plentiful development of a coarse, white, and later, brown mycelium with small brown sclerotia. The growth of the hyphae is of a peculiar radiating type, resembling densely crowded spokes of a wheel, and is often further characterized by concentric zones. Very often the mycelium, the sclerotia, and the paper wrapper form a thick, dry and hard mat, or the hyphae may hold the wrapper quite firmly to the fruit so that shreds of it remain when the fruit is unwrapped. Examination of decayed fruit in the market often reveals the original soil rot lesion with its firm, brown and concentrically marked center surrounded by water-soaked but firm tissue. Later there appears the coarse white mycelium which turns brown and forms sclerotia. The water-soaked areas are often referred to by the trade as "water blisters."

The fact that this rot is not limited to the blossom-end of the fruit, and is accompanied by a mycelium, differentiates it from blossom-end rot. It may follow blossom-end rot, however. Its solid mat of brown mycelium and its large lesions differentiate it from nail-head spot. The brown mat of fungus and the more regular and more closely arranged markings also differentiate soil rot from the buckeye rot. Furthermore, in the field, soil rot always shows these concentric rings while buckeye rot may not. Soil rot differs from Phoma rot by the absence of a black center with black pycnidia and from anthracnose by the absence of the orange-pink spore heaps.

Soil rot is especially severe on Florida tomatoes during rainy weather. It also occurs in early shipments of California tomatoes.

The original infection takes place from the soil in the field. Fruits touching the ground or hanging low enough to be spattered by the soil during rains are infected. The rot develops and spreads in the pack.

This disease can be controlled in the field by staking the plants. It is advisable to sort out diseased fruit in the packing house, since affected fruit is unmarketable, unfit for food, and a menace to sound fruit.

Ref. (56).

TOMATO, BLOSSOM END ROT

TOMATO: BLOSSOM-END ROT.

Cause: Non-parasitic (probably irregular water supply).

In the early stages this disease appears as a small spot at the blossom-end, or it may involve the whole blossom surface of the fruit. At this stage the spot looks like a bruise, is dark green in color and water-soaked. Later it becomes darker, and the affected tissue collapses and becomes firm and leathery. Sometimes the collapse is so sharply marked that the lesion appears as a distinct depression. Generally, however, it results only in a flattening of the end of the fruit. The lesions are very shallow and dry; this, together with the restriction of this injury to the blossom-end, serves to differentiate blossom-end rot from the buckeye rot of green tomatoes and the soil rot of both green and ripe fruits with which it might be confused.

Blossom-end rot is a field disease, and occurs in all tomato growing districts.

Affected tissue is predisposed to secondary rots, which develop in the field, in transit, and in storage. The non-parasitic injury is, in fact, commonly followed by fungous rots.

Blossom-end rot has not been successfully controlled. Regulation of the water supply, and the use of varieties suited to the soil and climate, are important points to consider.

It is not advisable to put badly affected fruit on the market. Slightly affected fruit, if not infected by fungi, can be used for food.

Ref. (3).

TOMATO MOSAIC.

TOMATO: MOTTLING.

Cause: Unknown, probably mosaic.

This disease is characterized by a more or less extensive brown discoloration of the surface of the fruit in which the affected areas show great diversity of pattern, such as circular spots, blotches, loops, and streaks. The affected areas are somewhat sunken and on green fruits the brown color may be absent. The affected tissue is firm and hard. The entire thickness of the wall and even portions of the septa (partitions) are involved. It seems that portions at least of the affected tissues are dead. At any rate, diseased fruits will not ripen properly in the ripening rooms, and the browned or sunken areas commonly become infected with various fungi. The disease resembles in some ways the mosaic disease of cucumbers.

In shipments of California tomatoes received in Chicago during the fall of 1918, this disease was undoubtedly the most important loss-producing factor, the loss amounting in some cases to 50 per cent or more.

No control is known. Since the symptoms are evident on the green fruits, it is advisable to sort out affected stock before shipment, as it is a total loss at destination.

TOMATO: BLACK ROT.

Cause: A fungus (Alternaria).

The most striking symptom of this disease is the conspicuous, black velvety growth of the fungus on the lesions. The disease generally occurs on ripe fruit, where it causes a slowly progressing rot. The fungus probably gains entry through wounds and growth cracks.

TOMATO - CAT FACE
GROWTH CRACKS (BELOW)

TOMATO: CATFACE.

Cause: Unknown, probably varietal.

In this disease the blossom end of the fruit is badly misshapen or puckered, probably due to the imperfect development of an area which at maturity is represented by an irregular leathery scar. Occasionally secondary rots occur in the malformed areas. The presence of catface in shipments lowers their market value.

TOMATO: GROWTH CRACKS.

Cause: Probably arrested and resumed growth.

These are rather deep ruptures or cracks radiating from the stem end. Occasionally they are arranged concentrically about the stem end. They seldom, if ever, occur in the blossom half of the fruit.

These cracks are actual wounds. Either before or after the fruit leaves the field, they are very likely to become infected with rot-producing organisms. From the market point of view, growth cracks assume a very great importance because rots so frequently start in them.

TOMATO RING ROT

TOMATO: RING ROT.

Cause: A fungus (Melanconium).

A large, circular, flattened lesion, very conspicuous because of its broad, concentric rings, is characteristic of this disease. The background color is brown and the center and the margin may be slightly elevated. Under moist conditions acervuli (singular acervulus), the fruiting bodies of the fungus, appear. The spores which exude from them frequently germinate and the resultant white mycelial growth covers the center of the lesions. The rotted tissue is firm, but affected fruits are worthless. Ring rot has been found to a limited extent in Cuban tomatoes.

Ref. (72a).

TOMATO

FUSARIUM ROT (ABOVE)
RHIZOPUS ROT (CENTER)
BLACK MOLD ROT (BELOW)

TOMATO: FUSARIUM ROT.

Cause: A fungus (Fusarium).

The differentiating symptom of this disease is a heavy, compact, pinkish-white growth of the fungus on the lesions. The advancing edge of the lesions frequently is free from visible fungous growth and is water-soaked and shrunken. The fungus probably attacks the fruit through wounds.

TOMATO: RHIZOPUS ROT.

Cause: A fungus (Rhizopus nigricans).

The general features of this disease have been taken up under the discussion of Rhizopus rot. It is a very soft, leaky rot. Affected fruit has an acid odor and when ripe usually has a bleached appearance. This rot rarely is found on green fruit.

The fungus attacks the fruit through breaks in the skin. These may be due to careless handling and so minute as to be invisible to the unaided eye, or they may result from skin pricks, nail injury, or mashing in packing or transit. The disease spreads in transit, the fungus growing through the wrapper from diseased to healthy fruit.

Ref. (56).

TOMATO: FOUL-SMELLING ROTS.

Cause: Bacteria and fungi.

The various tomato rots that are accompanied by foul odors have not been studied sufficiently to be differentiated and named.

INFECTED NOT INFECTED

TOMATO SUN SCALD.

TOMATO: SUN-SCALD.

(See Sun-scald).

TURNIP: GRAY MOLD ROT; BOTRYTIS ROT.

(See Gray Mold Rot).

TURNIP: SLIMY SOFT ROT; BACTERIAL ROT; SOFT ROT.

(See Slimy Soft Rot).

WATERMELON ANTHRACNOSE.

WATERMELON: ANTHRACNOSE.

Cause: A fungus (Colletotrichum lagenarium).

In its early stages, anthracnose is characterized by small circular raised welts on the rind, dark green in color. As these increase in size, the centers turn brown, become sunken, and under moist conditions the pink acervuli of the fungus are formed. As a lesion enlarges, the center becomes more sunken, often turns black, and eventually the surface cracks, exposing the rotted tissue or a cavity underneath. The fungus causes a rather slowly advancing dry rot of the rind tissue, and finally penetrates the edible pulp. Under extremely dry, hot conditions, such as occur in California during the melon harvest, the lesions may remain in the blister or nail-head stage.

Anthracnose is a serious disease of the vines and fruits in the field. The fungus is spread with the seed, overwinters in the soil, and spreads in the field during rainy weather. This is the same fungus which causes cucumber and muskmelon anthracnose.

Many fruits are rendered worthless in the field. The disease is also present in almost every car of melons shipped north as a result of field infection which may or may not have been detectable at the time of loading. The lesions enlarge during transit and not only seriously impair the appearance of the fruit, but open the way for the entrance of other rot-producing organisms.

Anthracnose is prevalent throughout the southern melon regions and the Ohio Valley. It is the source of great losses in the field, in transit, and in the market.

The disease can be controlled in the field by crop rotation, use of disease-free seed, and spraying with Bordeaux mixture.

Ref. (58).

WATERMELON STEM END ROT.

WATERMELON: STEM-END ROT.

Cause: A fungus (Diplodia).

This is a rapidly progressing soft rot of the stem end of the melon, marked externally by a brown discoloration and shrivelling followed by the development of abundant black pycnidia or a dark, gray, moldy outgrowth of the fungus. Whitish spore masses are often discharged from the pycnidia. The disease usually affects the stem-end of fruit, but occurs also at wounds and, in the field, as a blossom-end rot.

While the rot is rather common in the field, diseased fruits are not often shipped, and it seems quite likely that infection in the case of the stem-end rot found in the market takes place through the end of the stem after it is cut. The rot then develops with extreme rapidity during transit.

This is the most important rot of watermelons, and is rather general in southern grown stock, causing very heavy losses in some cars.

Stem-end rot as it occurs in transit can be prevented by proper treatment of the melons at the time of loading. After a tier of melons is in place, the ends of the stems are cut off and a paste made of starch and copper sulphate is applied to the freshly cut surfaces. This prevents infection by the fungus.

Ref. (42); (51).

WATERMELON: MISCELLANEOUS DISEASES.

BLOSSOM-END ROT.

Cause: Unknown (not parasitic).

This disease appears as a flattened, dry, leathery, blackened spot about as big as a silver dollar, at the blossom-end of the fruit. The tissues underneath are sound unless the killed area becomes infected with some fungus such as the Diplodia of stem-end rot, which is a common secondary invader.

While the cause of this trouble has not been demonstrated, it is quite likely that it is due to an unbalanced water supply as in the similar disease of tomatoes. Although of minor importance in the market, it may open the way to rot-producing fungi.

Ref. (51).

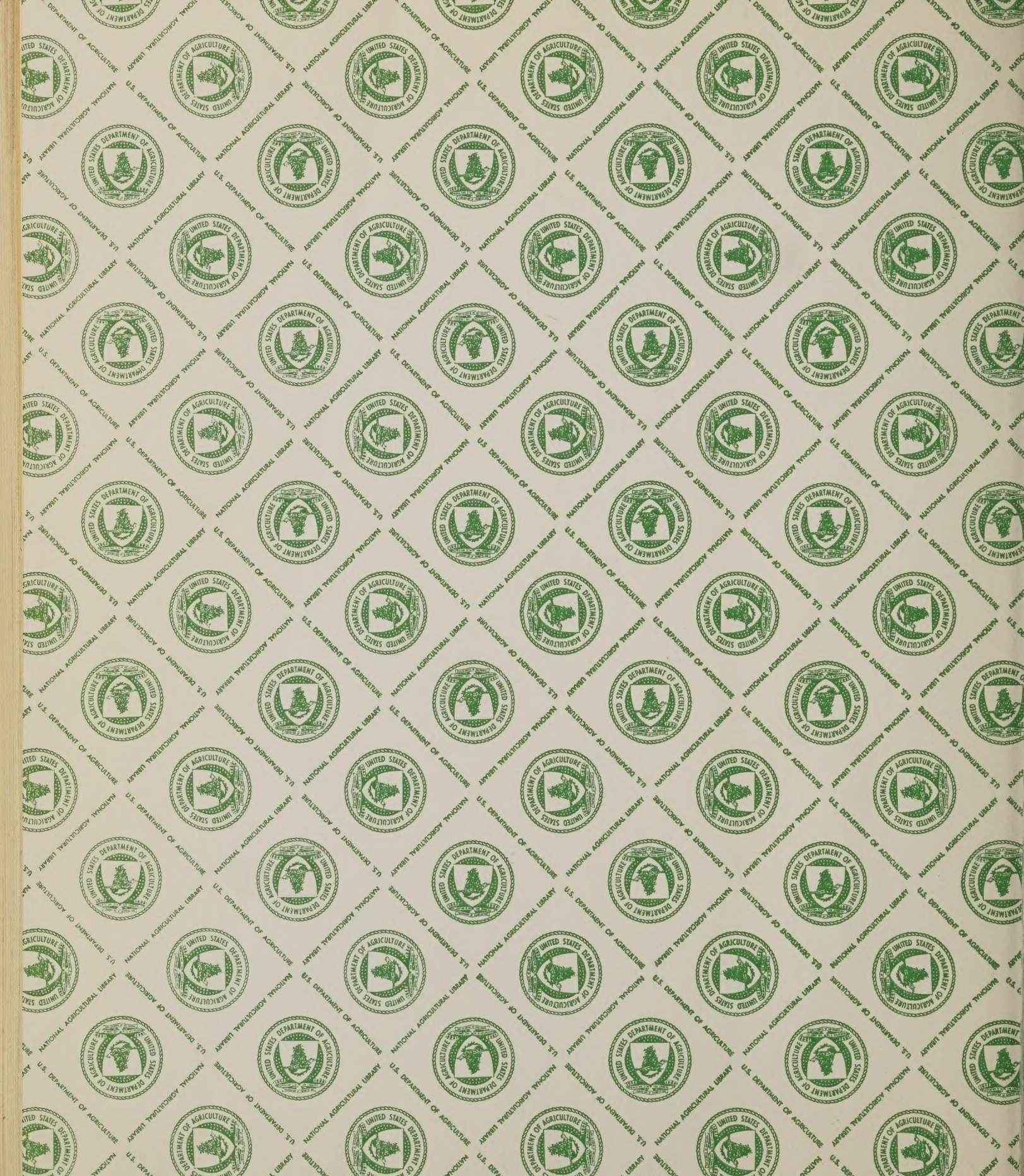
CHEMICAL INJURY AND ABRASION.

Cause: Contact with the walls or floors of cars.

This injury consists of large brown sunken areas or gouged channels in the rind. It is most common in cars previously used for chemicals such as fertilizers, especially where some shifting of the load has occurred. Sometimes fungi gain entrance through these injuries, and cause decay.

Compared with stem-end rot, this trouble is not of common occurrence.

Ref. (51).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